Simultaneous T_2- and T_1-mapping for cardiac applications

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Introduction: Cardiac MRI can depict areas of acute and chronic myocardial infarction (MI) by using different imaging sequences. MR determination of myocardial viability is typically performed by observing delayed enhancement (DE) of Gd contrast agent in necrotic tissue [1]. For improved contrast of such areas an inversion recovery sequence (T_1-weighted) is usually applied. For assessing the zone at risk a T_2-weighted sequence has to be applied [2]. The combination of both contrasts in one interleaved T_1- and T_2-weighted sequence can distinguish between: normal and infarcted myocardium; blood and MI [3]; and acute and chronic MI in one scan simultaneously. A quantitative analysis of the T_1 and T_2 relaxation times has the potential to prescribe standardized cutoffs between 'normal' and 'affected' myocardium and eliminate observer-input in delineation as well as determine the concentration of Gd inside of the myocardium. Because multipoint T_1 mapping techniques usually require a sufficiently long relaxation delay before each inversion pulse [4, 5], we propose an extension of a free breathing ECG-triggered inversion recovery sequence for T_1-mapping, where the relaxation pause is used to acquire images for a T_2-map. First results are shown in phantoms and in vivo in 7 healthy volunteers and a patient with an acute myocardial infarction (3 days after infarction).

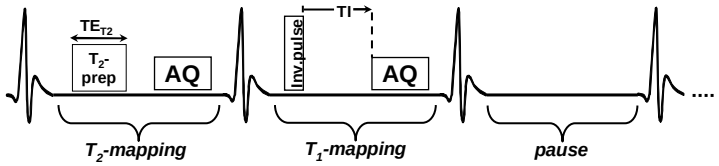


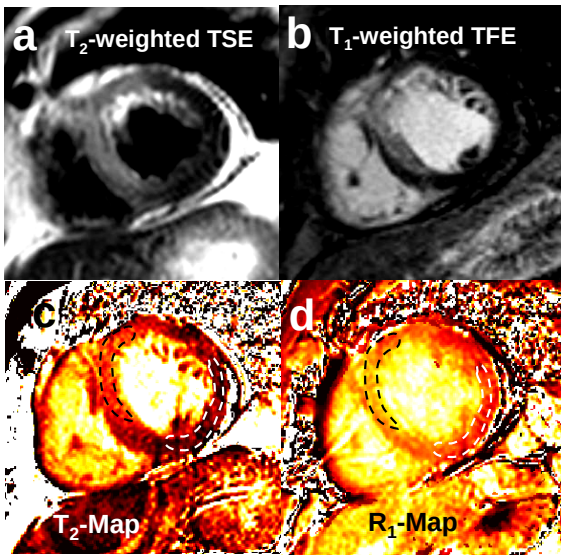
Figure 1: ECG-triggered interleaved T_2-T_1-mapping sequence

Phantom	T_1 (Ref.) [ms]	T_1 (interl.) [ms]	deviation T_1 (mean)	T_2 (Ref.) [ms]	T_2 (interl.) [ms]	deviation T_2 (mean)
1	421±5	445±4	+6%	55±1	52±3	-5%
2	400±6	423±4	+5%	92±1	91±1	-1%
3	727±14	669±12	-8%	110±1	103±2	-6%

Table 1: measured T_1 and T_2 relaxation times (phantoms)

Methods: Imaging has been performed on a clinical 1.5 T scanner (Achieva, Philips Medical Systems) using cardiac coil array (5-element). An ECG-triggered and navigator gated interleaved T_1- and T_2-mapping sequence was applied to quantify T_1 and T_2 of the myocardium. The proposed acquisition consisted of an interleaved 2D SSFP sequence with two different preparation pulses (Fig 1). The first interval contained a T_2-preparation pulse which was performed with four different echo times (TE_{T2prep}=20-160ms) and one without T_2-preparation in order to sample the T_2-relaxation curve. For the T_1-mapping in the second interval, an inversion recovery (IR) sequence with four variable time delays (TI=25-600ms) and one without the inversion pulse (to measure the full longitudinal magnetization) was used. The time delay TI between inversion pulse and image acquisition was varied by shifting the inversion pulse towards the QRS complex of the cardiac cycle, ensuring each image acquisition was at the same cardiac phase. Using this sequence we acquired 5 relaxation phases for each relaxation curve. In each interval 24 refocused gradient echoes were acquired (α = 60°, TR/TE = 3.7 / 1.86 ms, resolution: 1.37/1.37.8.00 mm^2, SENSE (AP): 2). T_1 was obtained using a three-parameter fit. Due to free breathing and different heart rates, the total scan time added up between 2 to 4 minutes. The results of the phantom and volunteer experiments were compared with results obtained with conventional mapping sequence [6] for the T_1 quantification and a spin-echo sequence for T_2-mapping as a reference.

Results: Table 1 illustrates the results of the phantom experiments, where is can be seen that the T_1 and T_2 values show good correlation with the relaxation times as measured by standard mapping sequences. For the volunteer study (N=7) the relaxation times of different human tissues were measured. The results also agree with values from literature, besides a systematic underestimation of long T_1 relaxation times. For the analysis of the patient data, two different regions of interest (ROI) were chosen to compare infarcted myocardium (black ROI) with healthy myocardium (white ROI). The T_2-map (Fig.2c) of the patient with an acute MI clearly depicts the area of the oedema with longer T_2-value as well as match with the bright region of the T_2-weighted image (Fig.2a). After contrast injection a T_1-weighted dataset was acquired to detect necrotic tissue (Fig.2b). The R1-map (Fig.2d) shows the corresponding quantification of T_1 values. In the area of delayed enhancement T_1 values are much shorter than before contrast injection. Furthermore a Gd concentration map was estimated from the difference of the R_1-maps before and after contrast injection (Fig.2) using the relaxivity of Magnevist r1 = 4.3 $mmol^{-1}s^{-1}$ [8]. The results show a much higher concentration of Gd in the area of necrotic tissue than in other parts of the myocardium.



	infarct (black ROI)	normal (white ROI	infarct (black ROI)	normal (white ROI)
before Gd [ms]	T_2 = 80±7	T_2 =40±6	T_1 =874±96	T_1 =739±80
after Gd [ms]	T_2 =81±8	T_2 =40±3	T_1 =314±16	T_1 =460±50
		Gd [mM]	c_1=0.473±0.03	c_2=0.14±0.04

Figure 2: short-axis view of a patient with acute MI, (a) depicts the edema and (b) the necrotic tissue (DE); (c) T_2-Map of corresponding slice; (d) R_1-map of corresponding slice after contrast injection; Table contains corresponding values for infarcted (black ROI) and normal tissue (white ROI)

Discussion and Conclusion: The free breathing navigator gated and ECG-triggered interleaved T_2- and T_1-mapping sequence can be applied to acquire quantitative maps of heart in only one scan without the need of registration. However by using this new sequence, a slight systematic underestimation of the absolute T_1 value can be observed. This is predominantly for tissue with long T_1 and short T_2 relaxation times; however the relative changes in T_1 correspond well with previously published results [7]. This requires further investigation and a correction algorithm for the calculated T_1 values. The method has been applied to differentiate between MI, blood pool and healthy myocardium. The method has the potential to differentiate between acute and chronic MI by estimating the concentration of Gd from ΔR1 in the necrotic tissue and to assess edema from T_2-maps.

References: [1] Kim RJ et al, Circulation 1996; 94:3318; [2] Abdel-Aty H et al, JMRI 2007; 26:452; [3] Kellman P et al, JMRI 2005; 22:605; [4] Messroghli DR et al, MRM 2004; 52:141; [5] Higgins DM et al, Med Phys 2005; 32:1738; [6] Look DC et al, Rev Sci Instr 1970; 41:250 ; [8] Rohrer M et al, Invest Radiol. 2005; 40;715; [7] Messroghli et al, MRM 2007; 58:34